POTENTIAL HEALTH IMPACTS RESULTING FROM BTGP NO_2 EMISSIONS

SYSAPP-83/094

April 1983

Prepared for

British Columbia Hydro and Power Authority
Environmental and Socio-Economic Services
Vancouver, British Columbia V6B 4T6

by

W. S. Simmons

Systems Applications, Inc.
101 Lucas Valley Road
San Rafael, California 94903

[64500-EF-81213r2]

CONTENTS

TABLES ii
INTRODUCTION 1
STUDIES IN HUMANS 3
CONTROLLED EXPOSURES 5
EPIDEMIOLOGY 8
CONCLUSIONS 9
REVIEW ARTICLES 14
REFERENCES R-1

TABLES

I	Selected Studies Using Animals	4
II	Summary of Effects Reported in Selected Human Studies	6
III	Effects on Pulmonary Function in Subjects Exposed to NO_2 and Other Pollutants	7
IV	Effects of Exposure tc NO_2 on Polumonary Function in Community Epidemiology Studies	10
V	Compilation of Effects Associated with Exposure to Nitrogen Dioxide in the Home in Studies Involving Gas Stoves	11

AN ANALYSIS OF POTENTIAL PUBLIC HEALTH EFFECTS DERIVING FROM NITROGEN OXIDE EMISSIONS FROM THE BURRARD THERMAL GENERATING PLANT

Introduction

Of the several nitrogen oxides which are known atmospheric contaminants (NO, NO_2, and NO_3) only one, Nitrogen Dioxide, presents a significant threat to public health. Nitric oxide (the nitrogen oxide which is actually emitted from combustion processes and is consequently referred to as a primary pollutant) is relatively non-toxic to humans compared to either nitrogen dioxide or nitrogen trioxide. Nitrogen trioxide (NO_3) is present in atmospheric environment in such small quantities and its residence time is so short that it is of concern only so far as its participation in the atmospheric chemistry processes which generate the atmospheric pollutant known collectively as photochemical oxidants. The process by which Nitrogen Dioxide is formed in the atmosphere from the primary emission, Nitric Oxide, is quite complex and requires, among other things, hydrocarbons, and ultra violet light energy. Atmospheric conversion of NO to NO_2 is highly dependent upon availability of O_3 and can occur at a rate as high as 70 percent per hour. The resultant concentration of NO_2 will be determined by atmospheric turbulence which controls plume dispersion. These considerations are of extreme importance in evaluating the potential health impact of Nitrogen Dioxide on the residents of the Burrard inlet communities for the following reasons:

> The Nitric Oxide emitted from the BTGP must be converted to Nitrogen Dioxide before it poses any significant hazard to the health of the resident population.

> In estimating the concentration of nitrogen dioxide to which the residents will become exposed, it is essential to correctly estimate the rate at which nitric oxide is being converted to nitrogen dioxide.

At the present time the United States Environmental Protection Agency enforces an annual primary ambient air quality standard (PAAQS) of 0.05 ppm (100 µg per cubic meter) for nitrogen dioxide, while the Canadian Federal Maximum Acceptable Ambient Atmospheric Concentration for nitrogen dioxide is 400 $\mu g/m^3$ averaged over one hour and 200 $\mu g/m^3$ averaged over 24 hours.

The Canadian Federal Maximum Desirable Concentration for an annual average is 60 $\mu g/m^3$.

In the course of the past three years several important comprehensive reviews of the biological activities of nitrogen dioxide and the adequacy of the existing guidelines and standards have been published. Shy et al. (1978) and Ferris (1978) have published excellent reviews of the effect of nitrogen dioxide on animals and humans and the United States Environmental Protection Agency (1979) has published an exhaustive analyses of all the effects of nitrogen oxide entitled Air Quality Criteria for Oxides of Nitrogen. Additionally, the National Research Council of the United States National Academy of Sciences published in 1977 a review of the medical and biologic effects of environmental pollutants (NAS, 1977) and the World Health Organization also in 1977 published Environmental Health Criteria No. 4, Oxides of Nitrogen. In summarizing the findings of these various documents, it is reasonable to conclude that, the U.S. EPA annual standard of 0.05 ppm or the Canadian Federal annual guideline of 60 $\mu g/m^3$, are adequate to protect public health. At the present time, health scientists within the U.S. Environmental Protection Agency, are debating:

- Whether or not a short-term primary ambient air quality standard for nitrogen dioxide is indicated.
- What concentration might be appropriate for a short-term nitrogen dioxide primary ambient air quality standard.
- What averaging time is appropriate for a short-term nitrogen dioxide PAAQS (i.e., the virtue of a one hour averaging time as opposed to a three hour averaging time).

It is not within the scope of this document to debate the scientific merit of the various studies which have gone into derivation of primary air quality standards for nitrogen dioxides. Presented here is a brief summary of the published findings reporting the effect of nitrogen dioxide on public health relevant to the petition of B.C. Hydro and Power Authority for permission to operate the BTGP on a continuous basis rather than part time as it is currently doing.

Studies in Animals

Animal studies, while useful in determining acute toxicity and pathological effects of inhaled toxicants, are of limited value in deriving PAAQS for humans due to the difficulty in interpreting the more subtle effects in terms that may be applied to humans. In spite of this limitation, however, animal studies are useful in determining what ultimate pathological effects a given inhaled toxicant may produce in human subjects.

The results of several recent studies on the effects of nitrogen dioxide on several species of laboratory experimental animals are summarized in Table I. Subtle biochemical responses of unknown significance to man have been reported to occur in rats and mice after three hour exposures to concentrations of nitrogen dioxide of approximately 0.2 ppm. A considerably more serious effect interpreted as an emphysematous type lesion has been reported in both mice and dogs exposed over six month periods to concentrations of nitrogen dioxide of 0.1 to 1.0 ppm nitrogen dioxide.

These animal studies while providing guidance in selecting biological end-points for study in human subjects are of little value in determining guidelines for safe human exposure because of the very large exposure dose utilized by the investigators.

Studies in Humans

Evidence derived from investigations of human response to nitrogen dioxide exposure falls into two broad categories: Those exposures conducted under carefully controlled clinical conditions in which small numbers of carefully selected human subjects are carefully monitored for respiratory and other physiological response and epidemiology studies in which large numbers of human subjects being exposed to naturally occurring concentrations of atmospheric pollutant are monitored over long periods of time. The controlled clinical exposure offers the advantages of specificity and precise knowledge of both dose and response, but is limited by the fact that only small numbers of subjects can be studied and only for short periods of time. The epidemiology study offers the advantage of large numbers of subjects being studied under real-world conditions, but provides only estimates of exposure concentrations and, for the most part, an essentially non-quantitative, subjective measurement of physiological response. In spite of their inherent weaknesses, when these

TABLE I. SELECTED STUDIES USING ANIMALS

NO_2 Concentration (ppm)	Exposure Duration	Species	Reported Effects	References
0.2	3 hours	Rat	Progressive inhibition in lung metabolism of PGE_2	Menzel (1980)
0.25	3 hours	Female Mouse	Increased pentobarbital induced sleeping time	Miller et al. (1980)
.64 NO_2 .25 NO	16 hrs/day 6 months	Beagles	Emphysematous changes	Hyde et al. (1978)
0.1 with 2 hr daily spike of 1.0	6 months continuous	Mice	Emphysematous changes	Port et al. (1977)
0.5 to 28	Continuous or intermittent 7 hr/day, 7 days/wk	Mouse	Increased mortality with increased time and concentration after exposure to infectious agent	Gardner et al. (1979)

two studies are taken together they provide the most valuable evidence available for determining what concentrations of a given atmospheric pollutant are least likely to produce adverse human health response in the general population.

Controlled Exposures

Summarized in Table II are the results of seven of the most recent and relevant controlled human exposures to nitrogen dioxide. Inspection of Table II reveals that healthy adults are quite resistant to relatively high concentrations of nitrogen dioxide for exposure periods up to approximately two hours. On the other hand, asthmatics and chronic bronchitics appear to be much more responsive to nitrogen dioxide insult. One study (Orehek 1976) reports that Carbachol* generated an enhanced increase in specific airway resistance in 13 of 20 asthmatics after an exposure to nitrogen dioxide at a concentration of 0.1 ppm for a period of one hour.

Another study (Kerr et al. 1979) reports that 7 out of 13 asthmatics experienced mild, transient symptomatic effects after exposure to 0.5 ppm nitrogen dioxide for a period of two hours.

In 1978 Folinsbee et al. reported that no physiologically significant changes in cardiovascular, metabolic, or pulmonary function could be detected in 15 healthy exercising adults after exposure to 0.6 ppm nitrogen dioxide for a period of two hours.

Based upon these studies, it would appear that asthmatics are somewhat more susceptable to nitrogen dioxide and further that the threshold for this type of response lies somewhere in the region of 0.5 ppm over a period of one to two hours.

Presented in Table III is a summary of several controlled clinical studies in which human subjects were presented with various concentrations of nitrogen dioxide in combination with other gases such as sulfur dioxide, ozone, and carbon monoxide. These studies are important in determining whether or not nitrogen dioxide interacts synergistically with other atmospheric pollutants in adversely affecting human respiratory function.

---

* Carbachol is a parasympathomimetic drug which is believed to act by releasing acetylcholine at certain vascular smooth muscle neuro-effector junctions and in sympathetic ganglia.

TABLE II. SUMMARY OF EFFECTS REPORTED IN SELECTED HUMAN STUDIES

NO_2 Concentration (ppm)	Exposure Durations	Study Population	Reported Effects	References
0.1 (188 $\mu g/m^3$)	1 hour	20 asthmatics	Specific airway resistance increased and effect of bronocho-constriction enhanced in 13 of 20 subjects after exposure to NO_2. Carbachol was used.	Orehek (1976)
0.5 (940 $\mu g/m^3$)	2 hours	10 healthy adults 7 chronic bronchitics, 13 asthmatics	7 asthmatics reported mild symptomatic effects.	Kerr et al. (1979)
0.5 to 5.0 (940 $\mu g/m^3$ - 9400 $\mu g/m^3$)	15 minutes	13 healthy adults 88 chronic bronchitics	Significant change in blood gases for healthy adults and bronchitics. No changes observed below 2.0 ppm	Von Nieding (1970)
0.6 (1130 $\mu g/m^3$)	2 hours	15 healthy exercising adults	No physiologically significant changes in cardiovascular, metabolic, or pulmonary function.	Folinsbee et al. (1978)
1.0 (1881 $\mu g/m^3$)	2 hours	16 healthy adults	No statistically significant changes in pulmonary function tests with exception of small changes in forced vital capacity.	Hackney (1978)
1.0 and 2.5 (1881-4700 $\mu g/m^3$)	2 hours	8 healthy adults	Increase in airway resistance at 2.5 ppm but not at 1.0 ppm	Bell and Ulmer (1976)
1.0 and 2.0 (1881 and 3762 $\mu g/m^3$)	2 and 1/2 hours	10 healthy adults	Alternating exercise and rest produced statistically significant decrease in hemoglobin, hematocrit, and erythrocyte acetylcholinesterase	Posin et al. (1978)

TABLE III. EFFECTS ON PULMONARY FUNCTION IN SUBJECTS EXPOSED TO NO_2 AND OTHER POLLUTANTS

Concentration (ppm)	Exposure Duration	Study Population	Reported Effects	References
0.05 NO_2 + .11 SO_2 + 0.025 O_3	2 hours	11 healthy	Increased sensitivity to bronchoconstrictor as shown by increases in R_{aw}. No effect on P_aCO_2 or R_{aw} without bronchoconstrictor.	Von Nieding et al. (1977)
0.50 O_3; 0.50 O_3 + 0.29 NO_2; 0.50 O_3 + .29 NO_2 + 30 CO	4 hours	4 healthy males	Minimal change in pulmonary function caused by O_3 alone. Effects not caused by NO_2 or CO.	Hackney et al. (1975)
0.25 O_3; 0.25 O_3 + 0.29 NO_2; 0.25 O_3 + 0.29 NO_2 + 30 CO	2 hours	7 males	Minimal change in pulmonary function caused by O_3 alone. Effects not increased by NO_2 or CO.	Hackney et al. (1975)
0.5 O_3; 0.5 O_3 + 0.5 NO_2	Rest 60 min. Exercise 30 min. Rest 30 min.	8 young adults	Response found only for O_3. Additive effect between O_3 and NO_2.	Horvath and Folinsbee (1979)

R_{aw} = Airway Resistance

P_aCO_2 = Arterial Carbon Dioxide Tension

.05 ppm NO_2 = 94 $\mu g/m^3$

0.29 ppm NO_2 = 546 $\mu g/m^3$

0.5 ppm NO_2 = 940 $\mu g/m^3$

A study reported in 1977 by von Nieding et al. suggests that a combination of nitrogen dioxide, sulfur dioxide and ozone caused an increase in airway resistance after a two hour exposure of 11 healthy subjects. However, Hackney et al. (1975) reported that a four hour exposure to various concentrations of ozone, nitrogen dioxide and carbon monoxide for four hours produced only minimal changes in pulmonary function which could be accounted for by ozone alone. In this study, no interactive effects were observed.

Horvath and Folinsbee in 1979 reported that a combination of 0.5 ppm ozone and 0.5 ppm NO_2 produced an additive but not synergistic response in the pulmonary function of 8 exercising young adults.

In summary, studies of nitrogen dioxide in combination with other gaseous pollutants suggests that nitrogen dioxide may interact with either ozone or carbon monoxide to produce an effect on human pulmonary function, but this interaction is apparently not synergistic and has been observed only at concentrations 2 to 3 times the NO_2 guideline fo 400 $\mu g/m^3$ for a 1-hour exposure.

Epidemiology

One of the most important early epidemiological studies of the effect of nitrogen dioxide on pulmonary function of human subjects has been referred to as the Chattanooga School Children Study and was reported by Shy et al. in 1970. Careful reexamination of this study has revealed serious problems with the method by which nitrogen dioxide was monitored in the environment and consequently the study is of little value in determining a safe concentration for human exposure. Numerous analyses of this earlier study have been published (NAS, 1977 and WHO, 1977) and it is now believed that the adverse response observed in the study group was due to suspended particulate matter and not nitrogen dioxide. It should be noted that this study served for some time as the basis for the current United States EPA PAAQS of 0.05 ppm nitrogen dioxide arithmetic annual average.

In 1976, Linn et al. reported on a study of office workers in Los Angeles and San Francisco . The median hourly concentrations of both nitrogen dioxide and photochemical oxidant were measured. While no differences in pulmonary function were observed in most tests, smokers in both cities demonstrated a greater change in pulmonary function than non-smokers.

Speizer and Ferris in 1973 studied the prevalence of chronic bronchitis in traffic policemen in urban Boston as opposed to patrol officers in less polluted nearby suburbs. No statistically significant excess of chronic respiratory disease was observed in the study groups. In the high exposure area, nitrogen dioxide peak 24-hour concentration was 0.055 ppm.

More recent and much more interesting epidemiological studies of both children and adults experiencing indoor exposure to nitrogen dioxide generated by gas cooking ranges are summarized in Table IV. These studies indicate that children, but not adults, who reside in homes where gas ranges are used to prepare food experience a higher incidence of respiratory symptoms and disease than those children that reside in homes where electric ranges are used for food preparation.

Speizer et al. (1980) report a significant association between history of serious respiratory illness before age two and use of gas stoves. In this study frequent nitrogen dioxide peaks of 0.4 - 0.6 ppm occurred in those homes utilizing gas ranges.

Melia et al. (1977) reported that bronchitis and asthma was increased in homes utilizing gas stoves. Unfortunately, nitrogen dioxide was not measured in the study homes at the time the study was executed.

In summary, the evidence presented in Table V is strongely suggestive that children are more sensitive to nitrogen dioxide than adults and that the possible threshold occurs at nitrogen dioxide concentrations of around 0.5 ppm.

Conclusions

The most recent and relevant studies into the effect on human health of nitrogen dioxide reveal that asthmatics, chronic bronchitics, and children under 12 years of age are more sensitive to nitrogen dioxide exposure than healthy adults. These data also indicate that in sensitive subjects the threshold nitrogen dioxide concentration for adverse health response in these sensitve subjects is approximately 0.5 ppm averaged over 1 hour. Studies have been published in which lower concentrations of nitrogen dioxide (0.1 ppm - 0.2 ppm) have been reported to produce an increased sensitivity in asthmatics to bronchoconstricting agents such as carbachol. However, the significance of these studies is not immediately obvious and it is not clear how they can be used

TABLE IV. EFFECTS OF EXPOSURE TO NO_2 ON PULMONARY FUNCTION IN COMMUNITY EPIDEMIOLOGY STUDIES

Exposure Concentrations (ppm)	Study Population	Reported Effects	References
Median hourly 0.07 NO_2 Median hourly 0.15 O_x Median hourly 0.35 NO_2 Median hourly 0.02 O_x	205 office workers in L.A. 439 office workers in San Francisco	No differences in most tests.	Linn et al. (1976)
High exposure area 24 hour high 0.055 NO_2 .035 SO_2 1-hour mean High exposure area 0.14 NO_2 to 0.30 NO_2 Low exposure area 0.06 NO_2 to 0.09 NO_2	128 traffic policemen in urban Boston and 140 patrol officers in nearby suburbs	No difference in various pulmonary function tests	Speizer and Ferris (1973) Burgess et al. (1973)

.055 ppm NO_2 = 103 $\mu g/m^3$
.06 ppm NO_2 = 113 $\mu g/m^3$
.07 ppm NO_2 132 $\mu g/m^3$
.09 ppm NO_2 = 169 $\mu g/m^3$
0.14 ppm NO_2 263 $\mu g/m^3$
0.30 ppm NO_2 564 $\mu g/m^3$
0.35 ppm NO_2 658 = $\mu g/m^3$

TABLE V. COMPILATION OF EFFECTS ASSOCIATED WITH EXPOSURE TO NITROGEN DIOXIDE IN THE HOME IN STUDIES INVOLVING GAS STOVES

Concentration (ppm)	Study Population	Reported Effects	References
Frequent peaks 0.4-0.6 (gas), maximum peak 1.0 (gas), 95th percentile 24 hour average in activity room 0.02-0.06 (gas) 0.01-0.05 (electric)	8,120 children, ages 6-10, 6 different cities	Significant association between history of of serious respiratory illness before age 2 and use of gas stoves. Small but statistically significant decreased in pulmonary function in children from gas homes	Speizer et al. (1980)
NO_2 not measured at time of study	2,554 children from homes using gas to cook compared to 3,204 children from homes using electricity, ages 6-11	Bronchitis, and asthma increased in homes with gas stoves	Melia et al. (1977)
NO_2 not measured in some homes studied	4,827 children, ages 5-10	Higher incidence of respiratory symptoms and disease associated with gas stoves	Melia et al. (1979)
Kitchens; weekly average 0.005-0.317 (gas) 0 006-0.188 (electric) Bedrooms: weekly average 0.004-0.169 (gas) 0.003-0.037 (electric)	808 children, ages 6-7	Higher incidence of respiratory illness in gas-stove homes	Florey et al. (1979) Goldstein et al. (1979)
Sample of households 24 hour average 0.005-0.11 (gas) 0-0.06 (electric) 0.015-0.05 (outdoors)	121 children, ages 0-5 346 children, ages 6-10 421 children, ages 11-15	No significant difference in reported respiratory illness between homes with gas and electric stoves in children from birth to 12 years old	Mitchell et al. (1974) Keller et al. (1979)
Peak hourly 0.25-0.50 (gas) maximum hour 1.0 (gas)	Housewives cooking with gas stoves compared to those cooking with electric stoves	No increase in respiratory illness	U.S. EPA (1976)
24 hour average 0.005-0.11 (gas) 0-0.06 (electric) 0.015-0.05 (outdoors)	Housewives cooking with gas stoves, compared to those cooking with electric stoves. 146 households	No evidence that cooking with gas is associated with increase in respiratory disease	Keller et al. (1979)
See Keller et al., 1979 for monitoring data	Members of 441 households	No significant difference in reported respiratory illness among adults in gas vs. electric cooking homes	Mitchell et al. (1974) Keller et al. (1979)

in deriving safe ambient air quality standards and guidelines for the protection of public health.

Dr. Benjamin Ferris (1978, p. 490) addressed the issue of a short-term primary ambient air quality standard for nitrogen dioxide and suggested:

> "Such a level might be 0.26 ppm (480 µg per cubic meter) for 1 hour, not to be exceeded 2 to 3 times per year."

This suggestion is consistent with the conclusion that the threshold concentration for adverse health response generated by nitrogen dioxide is approximately 0.5 ppm (940 µg per cubic meter) for approximately 1 hour. Assuming a margin of safety of a factor of 2, we may argue that a reasonable short-term standard for the protection of public health would be 0.25 ppm (470 µg per cubic meter) for 1 hour. This number is consistent with the Canadian Federal 1-hour Maximum Acceptable Level of nitrogen dioxide of 0.21 ppm (400 µg per cubic meter).

The review and analysis of measured NO_2 concentrations in the GVRD [Concord Scientific Corporation (CSC)] clearly show that nitrogen dioxide has not posed a threat to human health in this region for the years 1978-1981. No exceedances of the annual arithmetic mean maximum acceptable level or any of the tolerable objective levels have been recorded. One exceedance of the 1-hour maximum acceptable level was recorded (470 $\mu g/m^3$) at T7 (Anmore Elementary School, 12 October 1979), but even this concentration is well below levels demonstrated to produce an effect on human health.

In an attempt to determine whether or not the proposed alteration in the operating schedule of the Burrard Thermal Generating Plant will result in an unacceptable impact on the existing ambient atmospheric nitrogen dioxide concentration in certain crucial areas of the Burrard inlet, B.C. Hydro and Power Authority has undertaken modeling studies of the nitric oxide

emissions from the BTGP. These site specific studies* reveal the possibility that the Canadian Federal 1-hour Guideline of 400 µg per cubic meter occasionally may be exceeded. Based upon the results obtained in field studies, it is possible to conclude that the maximum 1-hour concentration that occasionally may be attained lies between 360 and 440 $\mu g/m^3$, a range not inconsistent with the maximum acceptable guideline of 400 $\mu g/m^3$.

Based upon our earlier observation that the threshold dose for nitrogen dioxide to produce an adverse health response in asthmatics or chronic bronchitics is approximately 0.5 ppm (940 µg per cubic meter), the existing Canadian Federal Guideline of 400 µg per cubic meter (averaged over 1 hour) contains a margin of safety somewhat greater than a factor of 2. This margin of safety insures that occasional excursion above the 1-hour guideline resulting from operation of BTGP (as indicated in the field studies*) will not result in an adverse impact on public health.

---

* See: Site Specific Air Quality Studies, page 14.

REVIEW ARTICLES

1. Benjamin G. Ferris, Jr., M.D., "Health Effects of Exposure to Low Levels of Regulated Air Pollutants--A Critical Review," J. Air Poll. Contr. Assoc., Vol. 28, No. 5 (May 1978).

2. Carl M. Shy et al. (1978), "Health Effects of Air Pollution," ATS News, Vol. 4, No. 2.

3. The National Research Council, "Nitrogen Oxides," in Medical and Biologic Effects of Environmental Pollutants, Washington, D.C., 1977.

4. World Health Organization, "Oxides of Nitrogen," in Environmental Health Criteria 4 (Geneva, 1977).

SITE SPECIFIC AIR QUALITY STUDIES

1. Analysis of Nitrogen Oxides Air Quality Data for the Greater Vancouver Regional District. Prepared for: British Columbia Hydro and Power Authority; Prepared by: Concord Scientific Corporation. February 1983.

2. Burrard Thermal Generating Plant Air Emissions and Impacts. Report No. ESS-39, A. E. Brotherston and J. H. Emslie. April 1982.

3. Greater Vancouver Regional District Ambient Air Quality Monitoring Data Report. 1981.

4. Vancouver Oxidant Study - Air Quality Analysis. Final Report. Prepared for: Environment Canada; prepared by: Concord Scientific Corporation. September 1982.

REFERENCES

Beil, M., and W.T. Ulmer (1976) "Wirkung von NO_2 in MAX-Bereich aud Atemmechanik and Acelytcholinempfindlichkeit bei Normal Personen," (Effect of NO_2 in Workroom Concentrations on Respiratory Mechanics and Bronchial Susceptibility to Acetylcholine in Normal Persons.) Int. Arch. Occup. Environ. Health, Vol. 38, pp. 31-44.

Burgess, W., L. DiBerardinis, and F.E. Speizer (1973), "Exposure to Automobile Exhaust. III. An Environmental Assessment," Arch. Environ. Health, Vol. 26, pp. 325-329.

Environmental Protection Agency (1976), "Scientific and Technical Data Base for Criteria and Hazardous Pollutants. 1975 ERC/RTP Review," EPA-600/1-76-023. US EPA, Research Triangle Park, North Carolina.

Florey, C. Du. V., R.J. W. Melia, S. Chen, B.D. Goldstein, A.G.F. Brooks, H.H. John, B. Craighead, and X. Webster (1979), "The Relation Between Respiratory Illness in Primary School Children and Use of Gas for Cooking, III. Nitrogen Dioxide, Respiratory Illness, and Lung Infection," International Journal of Epidemiology, Vol. 8, p. 347.

Folinsbee, L.J., S.N. Horvath, J.F. Bedi, and J.C. Delehunt (1978), "Effect of 0.62 ppm NO_2 on Cardiopulmonary Function in Young Male Non-Smokers," Environmental Research, Vol. 15, pp. 199-205.

Gardner, D.E., F.J. Miller, E.J. Blommer, and D.L. Coffin, " Influence of Exposure Mode on the Toxicity of NO_2," Environ. Perspec. (In press).

Goldstein, B.D., R.J. W. Melia, S. Chen, C. Du. V. Florey, D. Clark, H.H. John (1979), "The Relation Between Respiratory Illness in Primary School Children and the Use of Gas for Cooking, II. Factors Affecting Nitrogen Dioxide Levels in the Home," International Journal of Epidemiology, Vol. 8, p. 339.

Hackney, J.D., W.S. Linn, J.G. Mohler, E.E. Pedersen, P. Breisacher, and A. Rosso (1975a) "Experimental Studies on Human Health Effects of Air Pollutants, II. Four-hour Exposure to Ozone Alone and in Combination with Other Pollutant Gases," Arch. Environ. Health, Vol. 30, pp. 379-384.

Hackney, J.D., W.S. Linn, D.C. Law, S.K. Karuza, H. Greenberg, R.D. Buckley, and E.E. Pedersen (1975b) "Experimental Studies on Human Health Effects on Air Pollutants, III. Two-hour Exposure to Ozone Alone and in Combination with Other Pollutant Gases," Arch. Environ. Health, Vol. 30 pp. 385-390.

Hackney, J.D., F.C. Thiede, W.S. Linn, E.E. Pedersen, C.E. Spier, D.C. Law and D.A. Fisher (1978) "Experimental Studies on Human Health Effects of Air Pollutants, IV. Short-term Physiological and Clinical Effects," Arch. Environ. Health, Vol. 33 (4), pp. 176-181.

Horvath, S.N., L.J. Follinsbee (1979), "Effects of Pollutants on Cardiopulmonary Function," Report to U.S. EPA, EPA Contract No. 6802-1723.

Hyde, D. J. Orthoefer, D. Dungworth, W. Tyler, R. Carter, and H. Lum (1978) "Morphometric and Morphologic Evaluation of Pulmonary Lesions in Beagle Dogs Chronically Exposed to High Ambient Levels of Air Pollutants," Lab. Invest., Vol. 38, pp. 455-469.

Keller, M.D., R.R. Lanese, R.I. Mitchell, R.W. Cote (1979), "Respiratory Illness in Households Using Gas and Electric Cooking: I, Survey of Incidents, " Environmental Research, Vol. 19, pp. 495-503.

Keller, M.D., R.R. Lanese, R.I. Mitchell, R.W. Cote (1979), "Respiratory Illness in Households Using Gas and Electric Cooking: II. Symptoms and Objective Findings," Environmental Research, Vol. 19, pp. 504-515.

Kerr, H.D., T.J. Kulle, M.L. McIlhany, P. Swidersky (1979), "Effects of Nitrogen Dioxide on Pulmonary Function in Human Subjects: An Environmental Chamber Study," Environmental Research, Vol. 19, pp. 392-404.

Linn, W.S., J.D. Hackney, E.E. Pedersen, P. Breisacher, J.V. Patterson, C.A. Mulry, and J.F. Coyle (1976), "Respiratory Function and Symptoms in Urban Office Workers in Relation to Oxidant Air Pollution Exposure," Amer. Rev. Resp. Disease, Vol. 114, pp. 477-483.

Melia, R.J.W., C. Du V. Florey, D.S. Altman, and A.V. Swan (1977), "Association Between Gas Cooking and Respiratory Disease in Children," Brit. Med. J. , Vol. 2, pp. 149-152.

Melia, R.J.W., C.Du V. Florey, S. Chen (1979), "The Relation Between Respiratory Illness in Primary School Children and the Use of Gas for Cooking, I: Results from a National Survey," International Journal of Epidemiology Vol. 8, p. 338.

Menzel, D.B. (1980), "Pharmacological Mechanisms in the Toxicity of Nitrogen Dioxide and its Relation to Obstructive Respiratory Disease," in Nitrogen Oxides and Their Effect on Health, Di Duk Lee, ed. Ann Arbor Science, Ann Arbor Michigan, pp. 199-216.

Miller, F.J., J.A. Graham, J.W. Illing, D.E. Gardner (1980), "Extra Pulmonary Effects of NO_2 as Reflected by Pentobarbitol-induced Sleeping Time in Mice," Toxicological Letters,(Accepted for publication).

Mitchell, R.I., R. Williams, R.W. Cote, R.R. Lanese, and M.D. Keller (1974), "Household Survey of the Incidence of Respiratory Disease in Relation to Environmental Pollutants," WHO International Symposium Proceedings: Recent Advance in the Assessment of the Health Effects of Environmental Pollutants, Paris, June 24-28.

Orehek, J., J.P. Massari, P. Gayrard, C. Grimaud, and J. Charpin.(1976), "Effect of Short-term, low-level Nitrogen Dioxide Exposure on Bronchial Sensitivity of Asthmatic Patients," J. Clin. Invest., Vol. 57, pp. 301-307.

Port, C.D., D.L. Coffin, and P. Kane (1977), "A Comparative Study of Experimental and Spontaneous and Emphysema," J. Toxicol. Environ. Health, Vol. 2, pp. 589-604.

Posin, C.K., M.P. Clark, M.P. Jones, J.V. Patterson, R.D. Buckley, and J.D. Hackney (1978), "Nitrogen Dioxide and Human Blood Biochemistry," Archives of Environmental Health, November-December, pp. 318-324.

Speizer, F.E., E.G. Ferris, Jr., W.M.M. Bishop, J. Spingler (1980), "Respiratory Disease Rate and Pulmonary Function in Children Associated with NO_2 Exposure," American Review of Respiratory Disease, Vol. 121, pp. 3-10.

Von Nieding, G., H.M. Wagner, H. Krekeler, U. Smidt, and K. Muysers (1970) "Absorption of NO_2 in Low Concentrations in the Respiratory Tract and its Acute Effects on Lung Function and Circulation," Paper No. MB-15G, Presented at the Second International Clean Air Congress of the International Union of Air Pollution Prevention Assoc., Washington, D.C., December 6-11.

Von Nieding, G., H.M. Wagern, H. Krekeler, U. Smidt, and K. Muysers (1971), "Minimum Concentrations of NO_2 Causing Acute Effects on the Respiratory Gas Exchange and Airway Resistance in Patients with Chronic Bronchitis," Int. Arch. Arbeitsmed., Vol. 27, pp. 338-348, Translated from German by Mundus Systems for Air Pollution Technical Information Center, U.S. Mundus Systems for Air Pollution Technical Information Center, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.

Von Nieding G., H.M. Wagner, H. Lollgen, and H. Krekeler, (1977), "Acute Effects of Ozone on Lung Function of Men," VDI-Ber., Vol. 270, pp. 123-129, (In German)